NONINVASIVE IMAGING OF VENTRICULAR ACTIVATION - APPLICATION TO PATIENTS WITH WPW-SYNDROME

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Abstract-**Noninvasive activation time (AT) imaging using electrocardiographic (ECG) mapping data provides information about electrical propagation through the heart with a high spatio-temporal resolution. This study presents an attempt to clinically validate AT imaging in two patients with WPW-syndrome. We acquired 62-channel ECG mapping data during treatment in the catheter laboratory. The individual torso geometry was obtained by magnetic resonance imaging. Based on this data the AT map was estimated for the entire ventricular surface off-line for validation purposes.**

The AT maps were computed for a pacing protocol in the region of the right ventricular apex and for sinus rhythm beats prior and after successful pathway ablation. For paced beats the first onset activation was always found to be located in the apical region of the right ventricular endocardium. For beats in sinus rhythm prior to pathway ablation the location of the first onset of activation matched with the anatomic position of the ablation catheter during successful ablation. After the treatment this spot of early basal activation disappeared in the computed AT maps.

Thus, we conclude that noninvasive AT imaging from ECG mapping data presents a novel clinical tool for assessment of cardiac electrical activation in reference to the associated anatomy.

Keywords **- Activation time imaging, cardiac electrophysiology, inverse problem**

I. INTRODUCTION

For the treatment of patients with tachyarrhythmias in structural normal hearts the assessment of the cardiac electrical activation has obtained increasing interest [1]. During a standard electrophysiological study the time onset of activation can be determined in vivo by catheter intervention in a relatively small number of points only (e.g. [2]). The catheter-based electroanatomical mapping system CARTO™ (Biosense Webster Inc.) enables the invasive imaging of the activation sequence at the endocardial surface of any of the cardiac cavities [3]. However, only mapping of stable rhythms is possible by this system and this procedure is time consuming. The Ensite™ system (Endocardial Solutions Inc.) computes endocardial potentials from data recorded by a non-contact mapping catheter solving an inverse problem [4]. Here, in principle an inverse solution can be obtained also for single beat data. Recently, also the determination of "incisional activation times" has become possible. However, the reliability of this new tool for activation time imaging in clinical routine still has to be demonstrated. Furthermore, it is often difficult or even impossible to insert the mapping catheter into the left atrial or right ventricular cavity.

It has also been suggested that activation times can be determined non-invasively by computing an inverse solution from body surface potential mapping data [5]. Recently, the feasibility of this approach has been validated for the human ventricle in sinus rhythm by comparing the computed activation-time-map with a simultaneously recorded electroanatomical map [6]. In this study we will continue the validation process to the investigation of WPW-syndrome (Wolf-Parkinson-White-syndrome) [2]. The anatomical position of the accessory pathway is determined during conventional treatment in the catheter laboratory. This information is compared with the anatomic location of the first onset of activation in an activation time map computed off-line. The results will also be compared with the activation sequence computed for sinus rhythm after successful pathway ablation and for paced rhythms.

II. METHODOLOGY

A. Non-invasive activation time imaging

Details on the mathematical background of this approach can be found elsewhere [7,8]. Shortly, a piecewise homogeneous isotropic model of the individual patients torso geometry is build up applying the boundary element method. Major inhomogeneities such as the lungs and the blood masses are considered in the model. For this model the zero potential is defined by a Wilson terminal. Matrix deflation is adopted for obtaining an unique linear equation system [9]. Applying the bidomain model within the cardiac mass the transmembrane potential enters the source term. From this model a lead field matrix $\mathbf{L}$ can be computed which linearly relates the electric potential $\mathbf{\Phi}$ in all electrodes and the transmembrane potentials $\mathbf{V}_m$ in the boundary element vertices on the surface of the heart:

$$\mathbf{\Phi} = \mathbf{L}\mathbf{V}_m \tag{1}$$

For the structural normal heart one can take advantage of the a priori knowledge about the cardiac action potential time course. During depolarization the transmembrane potential rises quickly (within about 1ms) from its resting value (about –90mV) to the plateau level (about 10mV). This can be modeled by approximating the transmembrane time course in each source point by a step-like template function with unknown time of activation-onset. The resulting transmembrane potential matrix will be denoted by $\mathbf{V}_m(\boldsymbol{\tau})$. Here, $\boldsymbol{\tau}$ is the vector containing the activation times in all P primary source points, which have to be determined in the inverse problem. The nonlinear operator $\mathbf{F}$ relating the activation times $\boldsymbol{\tau}$ and the electric potential $\mathbf{\Phi}$ can be introduced by writing:

$$\mathbf{\Phi} = \mathbf{L}\mathbf{V}_m(\boldsymbol{\tau}) = \mathbf{F}(\boldsymbol{\tau}) \tag{2}$$

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Abstract

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The activation times $\boldsymbol{\tau}$ should be chosen in such a way that the associated forward solution fits the data. In other words $\|\mathbf{F}(\boldsymbol{\tau})-\boldsymbol{\Phi}\|$ should give zero, or in the presence of measurement noise and model error a relatively small value. Applying also Tikhonov regularization of second order for requesting spatial smoothness of the reconstructed activation pattern, one can define the following cost functional:

$$\Psi = \left\|\mathbf{F}(\boldsymbol{\tau})-\boldsymbol{\Phi}\right\| + \lambda^2 \left\|\Delta\boldsymbol{\tau}\right\| \rightarrow \min \qquad (3)$$

Here, $\Delta\boldsymbol{\tau}$ denotes an approximation of the surface laplacian of the activation times. Equation (3) can be solved by a deterministic optimization scheme. Note that this approach includes spatial as well as temporal regularization. The amount of spatial regularization is controlled by the regularization parameter. Temporal regularization is given by a template function for the transmembrane potential.

B. Study protocol

Two male subjects, 20Y (subject A) and 21Y old (subject B), with structural normal hearts and WPW-syndrome underwent radio frequency (RF) ablation of the accessory pathway. Before treatment in the catheter laboratory individual anatomical data was obtained by magnetic resonance imaging (MRI) using a Magnetom-Vision-Plus^{TM} 1.5T scanner (Siemens AG, Erlangen). Ventricular geometry was recorded in CINE-mode during breath-hold (expiration, 21×7 oblique short axis scans, 6mm spacing). The lungs and the torso shape were recorded in T1-FLASH-mode during flat breath-hold (expiration, 40 axial scans, 10mm spacing). 12 markers (vitamin E capsules, 7 anatomical landmarks on the anterior and lateral chest wall, 5 electrode position on the patients back) where used to couple all data acquired geometrically to the MRI frame. From this data a boundary element volume conductor model of the end-diastolic geometry consisting of about 2500 triangles (about 600 source points on the ventricular surface) were built up for each patient.

The patients were moved to the catheter laboratory and ECG mapping data was recorded during the diagnostic catheter study prior to radiofrequency ablation and in the waiting period after successful ablation of the accessory pathway. Electrocardiographic mapping data was collected in 62-channels by the Mark-8 system (Biosemi V.O.F.). A Wilson-terminal defined the reference potential. The sampling rate was 2048 Hz. Signals were bandpass filtered with a lower corner frequency off 0.3 Hz and an upper corner frequency of 400 Hz. The AC-resolution of the system is 500 nV/bit (16 bit per channel). Some example signal traces are shown in Fig. 1.

Radiotransparent carbon electrodes were used in order to allow simultaneous X-ray examination. The position of 52 electrodes on the anterior and lateral chest wall was digitized by the Fastract® system (Polhemus Inc.). Additionally the position of the 7 anterior and lateral landmarks was digitized in order to allow coordinate transformation to the MRI frame. The location of the 5 upper posterior electrodes was identical with the position of the 5 posterior MRI-markers.

III. RESULTS

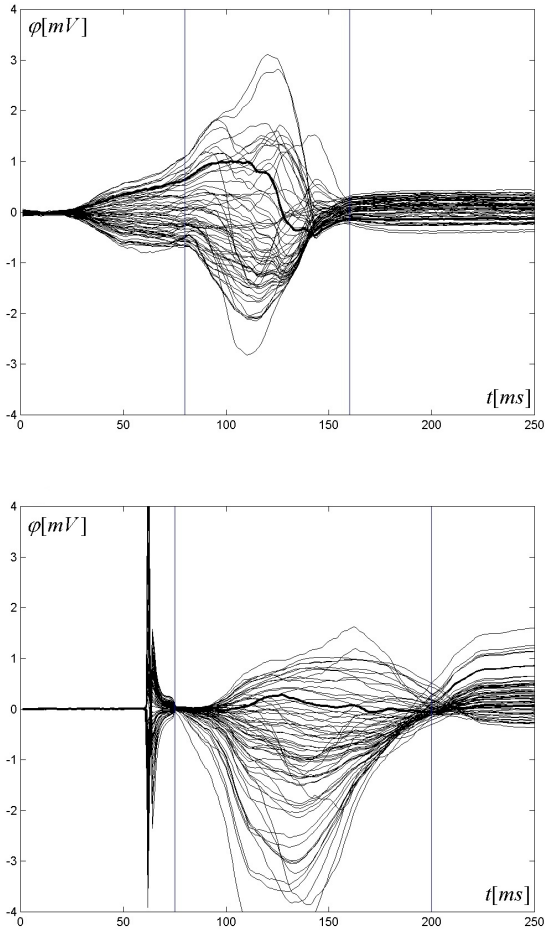

Fig. 1 ECG signal traces (65-channels) recorded in subject B are shown in a butterfly-plot. The upper panel depicts WPW-sinus-rhythm signals prior to pathway ablation. The lower panel depicts the signals from pacing in the right ventricular cavity. The bold line marks Wilson lead V_5. The plotted traces are single beat data. No signal processing except a baseline correction prior to the P-wave or stimulus was carried out.

Fig. 2 shows two activation sequences computed for subject A. For WPW-sinus-rhythm the earliest spot of activation was found at the base of the heart in a right anterior septal position. This finding agreed with the anatomic position of the radio frequency catheter during successful ablation of the accessory pathway in subject A. This spot of early activation disappeared in an activation sequence computed for sinus rhythm after pathway ablation.

For a beat stimulated by a diagnostic catheter in the apical region of the right ventricular cavity the first onset of activation matches with the anatomic position of the catheter tip. For the right ventricular free wall relatively early activation times were computed. In contrast for the left lateral wall late activation times were found.

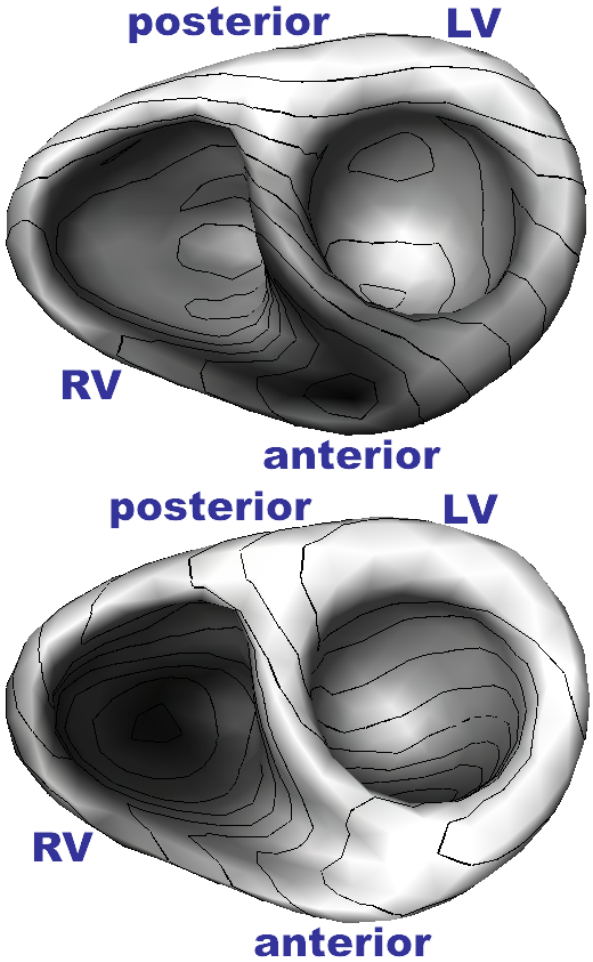

Fig. 2 Ventricular activation times computed for WPW-subject A are shown in gray scaling. Black marks early and white marks late activation. Isochrones are plotted in steps of 10ms. The ventricles are shown in a short axis view, looking from the base towards the apex. The upper panel shows the activation in WPW-sinus-rhythm before pathway ablation. The first onset of ventricular activation occurs at the base in a right anterior septal location (dark area). This location correlates with the position of successful ablation of the accessory pathway. The lower panel shows the activation sequence computed for a paced beat. A diagnostic catheter was positioned at the apical region of the right ventricular cavity for pacing. This location again corresponds with the area of first activation (dark area). Note that in this case the right ventricle (RV) is activated relatively early while the left ventricle (LV) is late. This leads to a desynchronized contraction of both ventricles.

For subject B in WPW-sinus-rhythm the first onset of ventricular activation is computed in the right-posterior part of the septal wall slightly below the base of the heart. This position fairly matches with the location of the RF catheter during ablation. For right ventricular pacing the first onset of activation is again found to be located in the apical region of the right ventricle. Again a remarkable time difference is found for the activation of the right and left ventricular free wall.

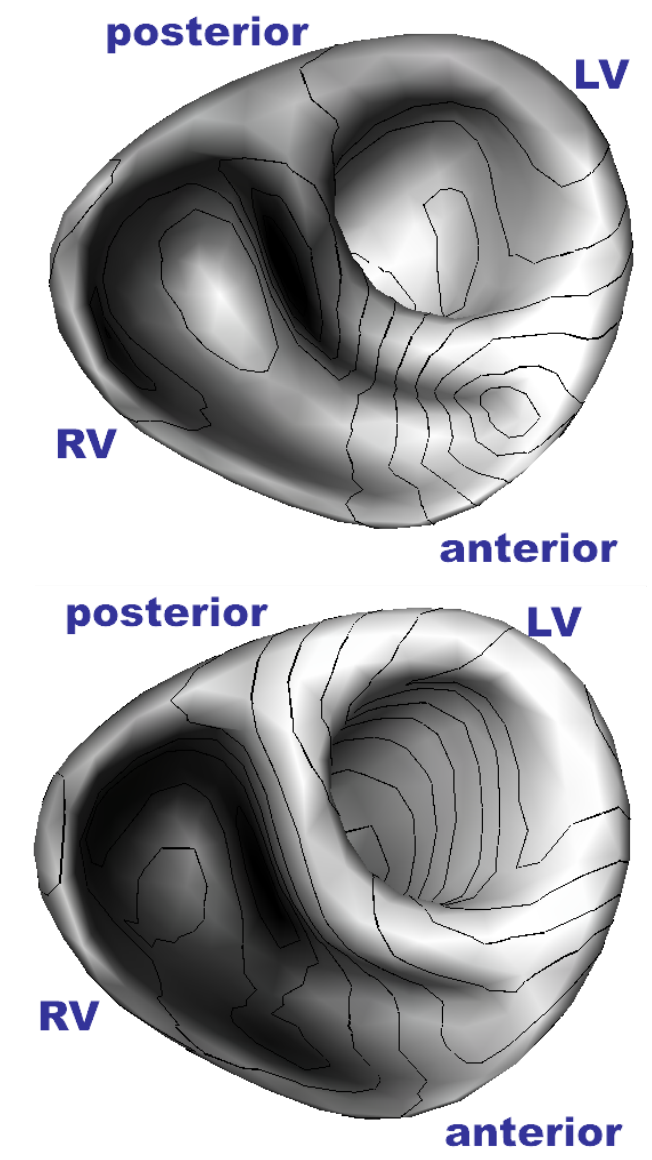

Fig. 3 Ventricular activation times computed for WPW-subject B are shown in gray scaling. Black marks early and white marks late activation. Isochrones are plotted in steps of 10ms. The ventricles are shown in a short axis view, looking from the base towards the apex. The upper panel shows the activation in WPW-sinus-rhythm before pathway ablation. The first onset of ventricular activation occurs slightly below the base in a right posterior septal location (dark area). The lower panel shows the activation sequence computed for right ventricular pacing. Similar as in Fig. 1 the first onset of activation is found to be in the apical region of the right ventricular cavity. Also in this patient a remarkable desynchronization of both ventricles is found.

IV. Discussion

The major scope of this study was the validation of ventricular activation time imaging by using anatomical information about the location of the first onset of activation. This information was obtained during treatment of WPW-patients in the catheter laboratory. The computed anatomical location of the spot of earliest activation was compared with the anatomical position of the RF-catheter or the pacing catheter in the right ventricle. Only qualitative estimation of error was carried out.

In both subjects for WPW-sinus-rhythm the computed spot of first ventricular activation was located at (or close to) the

heart base and its location fairly matched the anatomic position of the radio frequency ablation site.

For right ventricular pacing the first onset of activation was found to be located in the apical region of the right ventricular cavity, which agrees with the anatomical position of the pacing catheter. The right ventricular free wall displays early activation while the left lateral wall is activated late. As a consequence there will be a remarkable desynchronization in the contraction of both chambers. This observation seems to be of particular interest in the context of recent pacemaker developments enabling bi-ventricular stimulation. This feature enables a resynchronization of both patients resulting in a positive hemodynamic effect for the patient.

While in [6] a quantitative validation of the computed activation pattern in the right ventricular cavity was performed this study focuses mainly on the computation of the location of the first onset of ventricular activation. This location will be of particular interest in the treatment of WPW-patients and monomorphic ventricular tachycardia [10].

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